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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles related to periodontology that appeared during the year of 1944 are published in this year's issues.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided by contributing the material for this issue.

Reprinted from the Dental Cosmos, August, 1906

A Plea for Continued Effort in the Field of Dental Literature—[*Extract*]

By I. N. BROOMELL, D.D.S., *Philadelphia, Pa.*

. . . In 1846 a writer of considerable note made the statement that, "The gums are subject to several diseases peculiar to themselves, besides other affections which may be considered as the index of constitutional derangement." . . . "the nature of which is somewhat mysterious" . . . "the alveoli of the teeth have also their distinct diseases and are liable to be affected by diseases of the constitution." In the above we recognize symptoms which are now collectively called "pyorrhea alveolaris". We have succeeded in selecting a name for the condition, one which sounds scientific and elegant, but how much more do we know positively regarding the etiology of the affection? Perhaps no other subject in dental or oral pathology has received the same amount of attention in recent years as has this one. The literature upon the subject is voluminous and perplexing, so much so perhaps, that one in attempting to write further upon the subject feels almost guilty of adding to the confusion. But there should be no hesitation in doing so, because as the subject now stands there are at least three separate and distinct views regarding the cause of pyorrhea alveolaris, any one of which may be right; but likewise all may be wrong. They are—note the doubtful way in which they are spoken of—"constitutional of some kind, local of some kind, and infectious of some kind." While this subject appears almost worn threadbare, we should encourage and welcome further literature bearing upon the cause or causes of this very prevalent malady. Nor is the etiological factor regarding pyorrhea alveolaris alone unsettled. While a goodly proportion of dental literature has been devoted to the consideration of a rational treatment of the condition, to a very great extent the treatment of today differs but little from that employed several years ago, and perhaps with but little better results . . .

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FORTY-FIVE

Unusual Inflammatory Reaction of the Oral Mucosa*

By GRACE ROGERS SPALDING, D.D.S., *Birmingham, Michigan*
and BALINT ORBAN, M.D., D.D.S., *Chicago, Illinois*

A PATIENT, female, 46 years of age presented herself with an inflammatory reaction of the gingiva and oral mucous membrane on September 30, 1940. Ever since that time the patient has been under periodic observation and different methods of treatment were employed to free her of the annoying symptoms of her condition. These symptoms were extreme sensitiveness to highly spiced and acid foods; also to those of coarse texture. No definite diagnosis was ever made—tentatively and descriptively the term desquamative gingivitis was used.

CASE HISTORY PREVIOUS TO SEPTEMBER 30, 1940

(According to the patient's statement)—(G.R.S.)

The latter part of December, 1935, the patient first noticed an uncomfortable sensation in the gingival tissue of the right upper bicuspid and first molar area. In the patient's own words, "It felt like a pecan shell under the gum, and the tissue looked bright red." A dentist was consulted, but he advised the patient to "go home and forget it."

However, the red area began to spread, although it was confined to the labial and buccal surfaces of the gingiva. In November, 1937, another dentist was consulted. At different times, he used iodine and various aniline dyes. He advised the patient to brush the teeth vigorously "to stimulate the gums." These instructions were followed until the discomfort was unbearable. The affected tissues became sensitive to acid, spiced, and coarse foods, and were extremely painful at times, from a burning or "smarting" sensation. This was often localized, but in some instances, all of the affected tissues were painful. The dentist stated frankly that he was unable to diagnose the condition, but he nevertheless tried everything that he thought might give relief.

In October, 1939, a physician was consulted who gave the patient a complete physical examination, with radiographs and laboratory tests. The conclusion reached was that the patient had a vitamin C deficiency. Accordingly, vitamin C was prescribed and taken from October, 1939, to February, 1940, without

* From the Department of Research, School of Dentistry, Loyola University, Chicago College of Dental Surgery.

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apparent benefit. During this period, a rectal operation for hemorrhoids and polyps was performed by another physician. The operation was followed by a siege of colitis, lasting several months. While in the hospital, a dentist from a diagnostic clinic was called in for consultation about the mouth condition. He was puzzled by it, but left with the advice to the patient to "swish the mouth out thoroughly."

In February, 1940, the patient being excessively nervous, consulted still another physician. A bacteriological examination of the mouth was made and the diagnosis was Vincent's infection. An injection of an antisyphilitic drug was given. This was followed by a severe reaction in the form of urticaria medicamentosa. No further treatment was given and there was no improvement in the oral condition. In the meantime the dentist continued palliative treatment.

MEDICAL HISTORY SINCE MAY, 1940

In May, 1940, a fourth physician was consulted who has had the case under direct supervision ever since. Several complete blood examinations have been made, but the findings have offered no diagnostic clues. A complete radiographic examination of the digestive tract has been made. Estrogenic hormone and vitamin treatment has been continuous from May, 1940, up to the present time. Other internal medication has been used, as well as a single blood transfusion as an experiment, and without results.

No permanent change in the oral condition was noted until about nine months ago, when the recurring attacks of severe discomfort became noticeably less frequent.

In May, 1944, the patient consulted a specialist in allergy diseases, to get relief from hay fever and asthma. Since that time she has had shots every five days and has been free from both these diseases.

DENTAL HISTORY SINCE SEPTEMBER, 1940

The first clinical oral examination of this case (by G.R.S.) showed a mouth with all teeth present excepting the third molars. All teeth were vital. Radiographic examination showed but little alveolar resorption. The gingival tissues in certain areas were *bright* red and highly inflamed. The condition was confined almost exclusively to the labial and buccal surfaces, the exception being the lingual surfaces of the gingivae around the lower first and second molars including the interproximal tissue. The gingival tissue over the upper left lateral and one-half of the approximating central and cuspid was practically normal. In some locations a whitish film covered the tissue, especially in the upper and lower right bicuspid and molar buccal area.

It was soon discovered that all of this abnormal tissue was exceedingly sensitive to any form of medication. However, to establish and to maintain a hygienic condition of superior quality seemed mandatory, regardless of the etiology. A careful and thorough prophylaxis was given weekly for eight weeks and monthly for four months, at the same time suggesting hygienic procedures to the patient who was most co-operative. Various mouth washes were tried, but all proved irritating, including a mild saline solution.

The patient's physician was consulted and it was concluded that the etiology was probably systemic. The patient was passing through the menopause and was being given every aid and careful, painstaking observation. Estrogenic hormone therapy was being continued.

Because the patient's health was improving under this therapy, it seemed logical to try the local application of an estrogenic ointment as had been described by Ziskin*, and reported successfully used by Yeretsky** for desquamative gingivitis. Thirty-three applications of the ointment were made according to the technique used by Ziskin at that time, (June-September, 1941) with no positive signs of improvement. The treatment was then discontinued.

The patient's home care consists of using wood points for cleaning the tooth surfaces next to the gingival margins and interproximally. English precipitated chalk is used with a soft toothbrush twice daily. A very mild solution of sodium bicarbonate is used as a mouth wash, being the only liquid which is non-irritating.

It was found that at the periods of extreme discomfort, acri-violet gave relief without subsequent irritation. This suggested a fungus infection. A bacteriological examination of the mouth, made with this thought in mind, revealed the presence of *Monilia albicans*. Systemic treatment seemed at that time to be contraindicated on account of other such treatment the patient was receiving. An effort was made to see

* J. D. Res. 18(4): 329-341, Aug., 1939
** J.A.D.A. 30(9): 729-737. May, 1943.

if an improvement could be accomplished by local measures only. Lugol's solution of iodine was applied at the time of a weekly prophylaxis (for five weeks), and by the patient in specified locations. The patient used it as a mouth wash ten drops in one-half cup of water, but later decided that it was irritating and discontinued its use. It has been used ever since at the time of prophylaxis treatments.

In August, 1944, the patient visited a relative who is a periodontist and who examined her mouth. He diagnosed the condition as "pyorrhea" and proceeded to treat an area in lower right incisor, cuspid, and bicuspid region, by very painful and radical subgingival curettage. The tissue involved did not return to its original condition until the following spring, which convinced us that the conservative method we had followed had been a wise one. There is no improvement in the section treated, much discomfort was experienced over several months, and the labial gingiva of the lower right cuspid, which was the slowest in healing, has receded more than one-eighth of an inch.

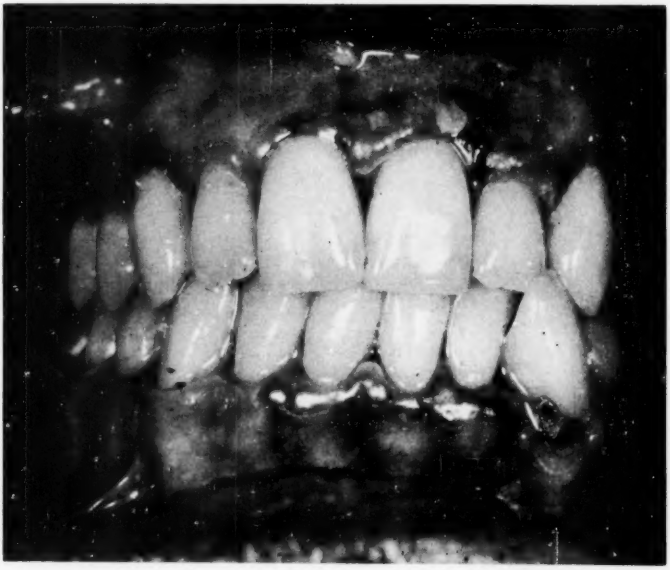

Fig. 1. View of oral mucous membrane of patient at time of biopsy.

HISTOPATHOLOGIC INVESTIGATION *(B.O.)*

In October, 1944, biopsy specimens were taken from the upper as well as lower jaws. At this time the oral mucosa appeared as shown in Fig. 1. The entire labial and buccal gingiva as well as mucosa was bright red—covered in limited areas by a whitish, milky film. This whitish film was heavier in some regions and could be removed by lifting it up with a pair of pliers. The entire mucosa appeared rather thin, transparent, fine in texture. In some areas there was a slight swelling of the gingiva. The biopsy specimens were taken from the right upper and lower bicuspid regions. The buccal part of the gingival papilla was removed with some of the vestibular mucosa.

The section of the biopsy specimen from the upper jaw is seen in Fig. 2. The gingival portion of the specimen shows the characteristic and usual signs of chronic inflammation. Plasma cell accumulation appears in the deep tissue layers and leucocytic infiltration is seen close to the gingival pocket, in the superficial tissue areas. The epithelium becomes thinner as the bottom of the gingival pocket is approached. The thin epithelium of the pocket is also infiltrated by leucocytes.

Of special interest in the present case is the inflammatory reaction in the vestibular mucosa adjacent to the gingiva. In this region, under normal conditions, the epithelium shows a regular stratified squamous appearance without hornification and with a gradual flattening of the surface cells; the

lamina propria is thin, fibrous, has a regular papillary layer and is free of inflammatory cell infiltration. In this case the epithelium shows no peg formation as a result of atrophy of the epithelium. In many areas the surface is extensively hornified. The hornification is characterized by the formation of several layers of flattened cells, showing only shadows of former nuclei, or empty spaces. Toward the cellular part of the epithelium a more homogenous layer—a stratum lucidum, is seen in the stratum corneum. Under normal conditions a stratum lucidum is never found in the oral cavity, even in the otherwise well hornified areas of gingiva and hard palate. In the present specimen, the stratum lucidum was well developed in localized spots, especially where the hornificatic was extensive. (Fig. 3A.) Wherever the hornification is heavy, a well-developed stratum granulosum can be seen.

The epithelium is considerably thinner than normal. The stratum spinosum consists of only 5-6 cell layers and the intercellular spaces are wider than normal. The stratum germinativum, the innermost layer, is not as regular as under normal conditions and wandering cells have invaded the epithelium. In some areas the hornification is not as well developed (Fig. 3B), and the granular layer is also thin. The entire epithelium consists of only a few cell layers and the border line of epithelium and connective tissue has disappeared. The cells of the germinative layer lost their cuboidal shape and regular arrangement; they can only vaguely be detected. The epithelium is badly disarranged and the intercellular spaces have been invaded by large numbers of lymphocytic elements.

The subepithelial connective tissue is densely infiltrated by inflammatory cells, mainly of the lymphocytic type (Fig. 3A and B). A great variety of cells in a rather haphazard arrangement can be detected in this chronic inflammatory granulation tissue. Most frequent are the lymphocytes and plasma cells, but peculiarly large mononuclear cells, of the macrophage type, and some "epitheloid" cells, can also be seen. Some cells appear in groups and form multinucleated giant cells of peculiar types (Fig. 4A and B). In certain areas some of the specimens have shown tissue decomposition, necrosis (Fig. 4A), while in others, no such necrosis could be observed.

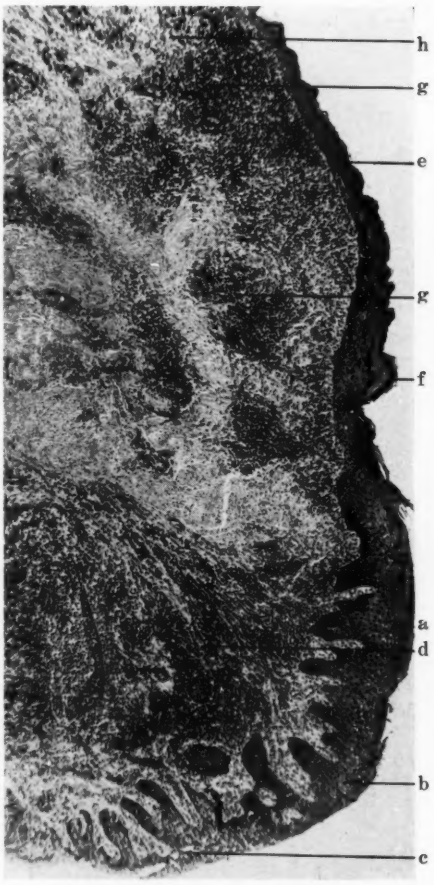

Fig. 2. General view of biopsy specimen.
a: gingival epithelium.
b: gingival margin.
c: epithelium of the gingival pocket.
d: chronic inflammation of the gingiva.
e: atrophic epithelium of the vestibular mucosa.
f: excessive hornification.
g: areas of inflammation where most giant cells were found.
h: area where lymph follicle (See Fig. 6) was found (in other sections).

In the deeper tissue regions, giant cells with large cytoplasmic bodies could be found, mostly in groups of 2 to 4 (Fig. 4C) surrounded by lymphocytes, plasma cells, and macrophages. Some of the giant cells create the impression that they are of the Langerhans type, which is typical for tuberculous granulation tissue. The nuclei in these cells were arranged on the periphery of the large cytoplasmic body (Fig. 4D).

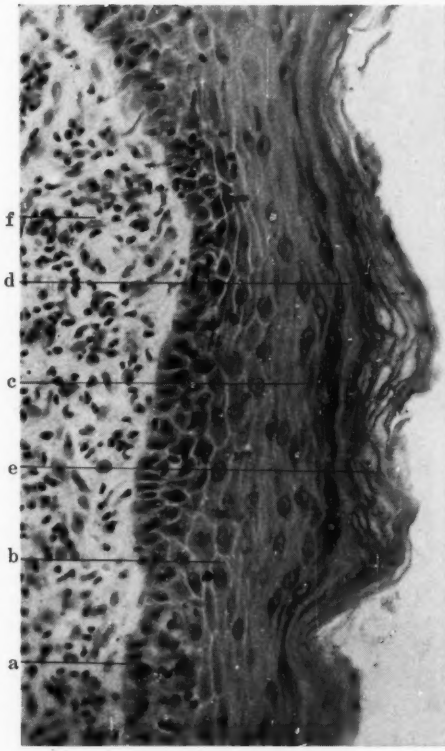

Fig. 3A. Extensive hornification of the atrophic epithelium of the vestibular mucosa.
a: germinative layer.
b: prickle cell layer (str. spinosum).
c: heavy granular layer.
d: stratum lucidum.
e: hornified layer (str. corneum).
f: chronic inflammation in the lamina propria.

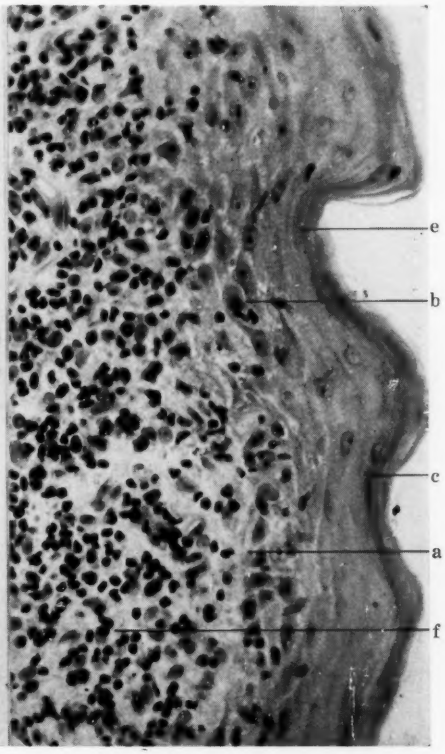

Fig. 3B. Atrophic epithelium of the vestibular mucosa. The border line of epithelium and connective tissue disappeared. (a).
b: wandering cells invade the epithelium.
c: thin granular layer.
e: thin hornified layer.
f: chronic inflammation with lymphocytes, plasma cells, and macrophages.

In several sections of this specimen, crystalline foreign bodies could be found within giant cells. (Fig. 5.) The crystalline substance can easily be observed with polaroid filters (Fig. 5B). Around the giant cells, frequently a large number of eosinophile leukocytes could be seen (Fig. 5A). Some eosinophile granules lie free in the tissue most probably as a result of a breaking-up of the eosinophilic leukocytes.

This type of granulation tissue, with the great variety of its cellular elements, and especially the presence of different types of giant cells, has never been observed in gingival inflammation.

There is another feature of this specimen which makes it different from others, i.e., the presence of a lymph follicle with an active germinative center (Fig. 6). This lymph follicle was located close below the epithelium, at the vestibular end of the biopsy specimen (Fig. 2h). It was seen only in about half of the sections obtained from the biopsy specimen of the upper jaw. With the exception of the lymph follicle, the findings were identical in both biopsy specimens from upper and lower jaws. In the germinative center of the follicle, a large number of mitotic figures could be seen, most probably within the large lymphoblasts. (Fig. 6B.) In no other biopsy specimen of the gingiva was a lymph-follicle ever observed or reported. All these findings in the epithelium, as well as in the connective tissue, indicate a chronic inflammatory reaction of an unusual type.

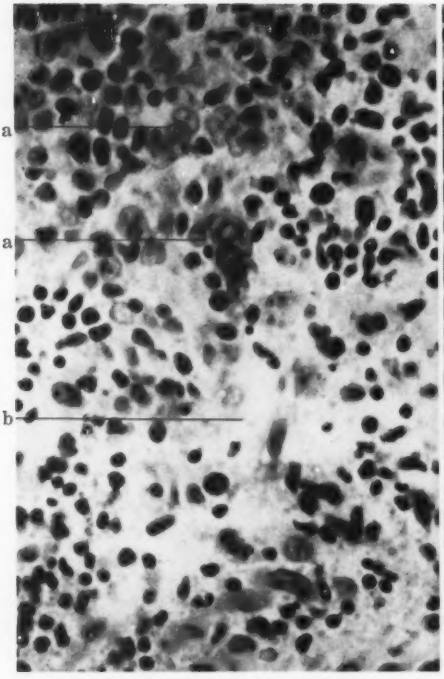

Fig. 4A. Giant cells (a) surrounded by lymphocytes. Necrotic tissue (b).

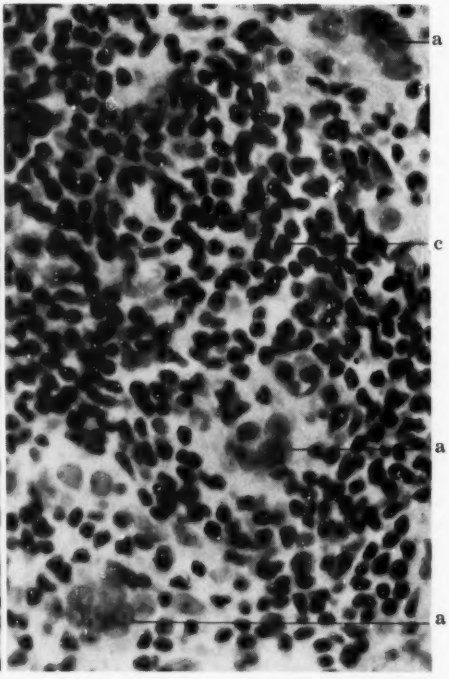

Fig. 4B. Giant cells (a) surrounded by lymphocytes (c).

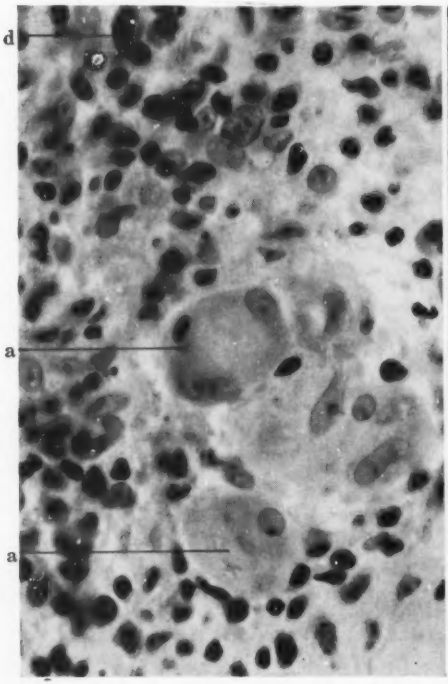

Fig. 4C. Giant cells (a) surrounded by lymphocytes and plasma cells (d).

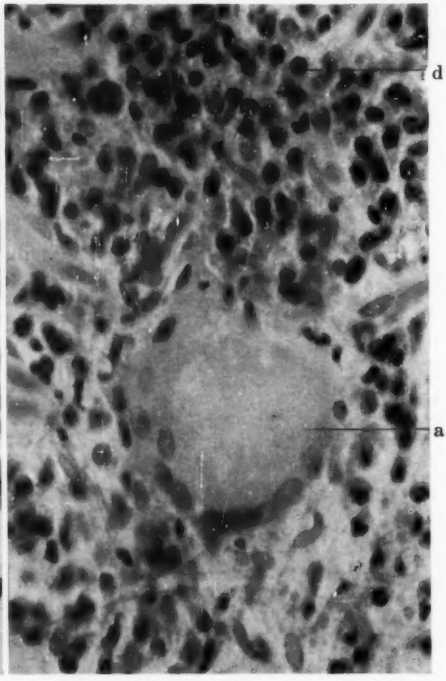

Fig. 4D. Giant cell (a) Langerhans type. Plasma cells (d).

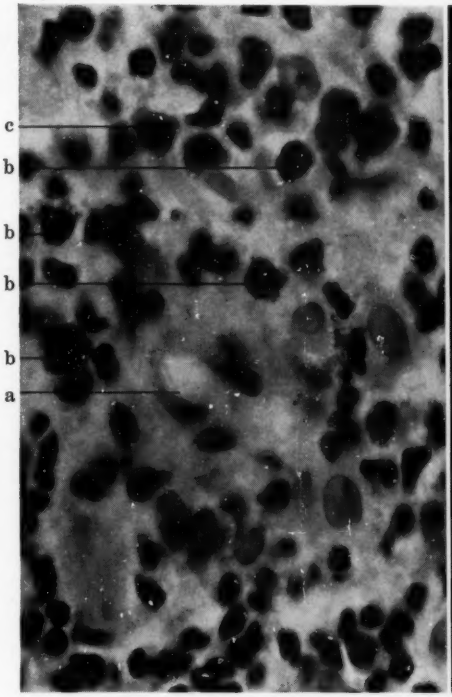

Fig. 5A. Giant cell with crystalline foreign body (a). Large number of eosinophile leukocytes (b). c: plasma cell.

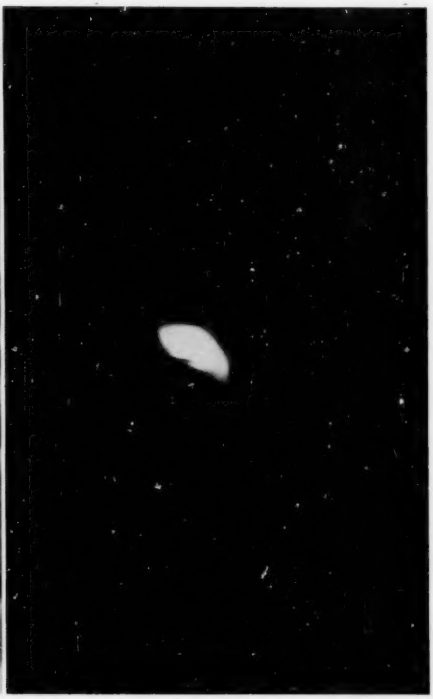

Fig. 5B. Same area photographed with polaroid filters to show crystalline body.

DIAGNOSTIC INTERPRETATION

The fact that this inflammatory condition was of such long standing, was so resistant against most attempts of therapy, and combined with the unusual appearance of the inflammatory reaction, made a clear-cut diagnosis difficult. Several pathologic conditions which are often manifested by skin symptoms had to be considered. These might have oral lesions, or only oral lesions such as lichen planus, mycosis fungoides, lupus erythematosus, syphilis, lymphogranulomatosis (Hodgkin's disease) or some fungus infection. To be able to differentiate between these conditions several experts in skin pathology, as well as general pathology, were consulted. Our sincerest thanks is due to all who gave us their time and valuable advice.

Lee McCarthy, author of the atlas of *Histopathology of Skin Diseases** and at present Commander in the USNR, studied the slides and showed them to several general pathologists. He claims that from a dermatological point of view, lichen planus, lupus erythematosus, mycosis fungoides and Hodgkin's disease could be ruled out. He believes that this condition represents a long-standing chronic inflammatory process with no specificity. In other words, no signs of malignancy, tuberculosis, syphilis, or actinomycosis. He agreed with the general pathologists, who saw the specimens, that this condition represents a chronic granuloma with numerous foreign body giant cells.

Lloyd W. Ketron of the Skin Dispensary of Johns Hopkins Hospital in Baltimore examined the specimens, but could not arrive at any specific diagnosis. He believes that this specimen shows the picture of a chronic inflammatory reaction probably to

* McCarthy, Lee. *Histopathology of Skin Diseases*. C. V. Mosby Co., St. Louis, 1931.

Fig. 6A. General view of lymph follicle (a). Close below the atrophic epithelium (b).

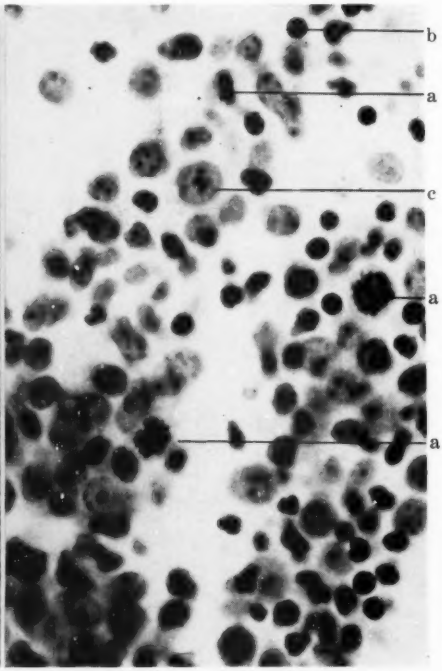

Fig. 6B. High magnification of the germinative center of the lymph follicle.
a: mitotic figures of lymphoblasts. Lymphocytes (b), lymphoblasts (c).

some infection. The possibility of lichen planus or lupus erythematosus would have to be considered, according to Ketron, but here the clinical aspect would be more important than the histologic. Ketron also expressed his opinion that lymph follicles appear occasionally in any chronic inflammatory process and, therefore, should not be given too much significance in this case. He found as the most interesting aspect, the presence of multinucleated cells. These he considered to be of the macrophage type.

M. Oppenheim, Professor of Dermatology at the Chicago Medical School ruled out lichen planus, lupus erythematosus, mycosis fungoides and lymphogranulomatosis. Dr. Oppenheim demonstrated a large number of specimens of these diseases and explained the different diagnoses.

E. Loeffer, Chief Pathologist of Cook County Hospital came to the conclusion that this specimen represented an unspecific chronic inflammatory condition.

CONCLUSION

After receiving these opinions, it was clear that we would not be able to make a definite diagnosis of this case even after a histopathologic investigation. There is no question about the fact that we deal with a chronic inflammatory condition. Syphilis had to be ruled out on the basis of repeated serologic investigations, which, in every instance, were negative.

There is one more feature in the history of this case which has to be considered, namely, the fact that a year ago bacteriologic tests of the oral flora showed the presence of a fungus *Monilia albicans*. In addition to this finding, the clinical observation was made that the use of acri violet made the mouth most comfortable and seemed to best control the inflammatory symptoms. It is known that *Monilia albicans* can produce inflammatory reactions known as moniliasis, thrush or soor, in the oral cavity. In this disease, the oral mucous membrane appears to be covered by a white membrane of varying density. It

adheres to the base; the base is red and bleeds upon removal of the membrane. It is mostly found in young poorly nourished individuals. The histopathological picture shows a chronic or sub-acute inflammatory reaction with focal areas of necrosis. Giant cells are not infrequent; monocytes and lymphocytes are most abundant. An excellent discussion of the nature of moniliasis, the clinical and bacteriologic picture, was published by A. R. Woodburne. *

It is very likely that in the present case a monilia infection is or has been active in producing the chronic and multiform inflammatory reaction.

MEDICAL REPORT—September, 1945

By H. B. STEINBACH, M.D.

Detroit 26, Michigan

THIS patient has been of considerable interest since all physical findings have been normal. Gastric analysis, pancreatic and liver function, stool, sugar n.p.n. blood chemistry, and serum globulin and albumin have all been within normal range. However, two facts were important: besides the local condition, the patient was a typical glandular type at an age that would fit the picture; second, she was an allergic asthmatic type.

Glandular therapy (theelin), which she had been taking before I saw her, gave her systemic relief. This I continued over a period of time. The gums and membrane remained spongy and carried a low grade inflammatory appearance and a tendency to bleed.

When placed on testosterol because of an intestinal upset, which frequently occurred particularly associated with menstrual periods, the gums improved as well as the intestinal symptoms. Following, there was an aggravation of menopausal symptoms. These have again been relieved by theelin. Since the patient has been confined to testosterol therapy and all other female glandular hormone's discontinued, the patient's gums have definitely improved. Her intestinal symptoms (spastic bowel, etc.) are now eliminated.

About the same time this latter therapy was begun, the patient was started on allergy therapy. This may have been a factor in her improvement.

There are, in my opinion, certain fundamental things which stand out in all low grade infections. Some of these have not been generally accepted:

1. There is a relation between glandular and allergic changes.

2. There is a definite colloidal and chrystaloidal chemical metabolism of cells, which make the above allergy and glandular relations.

3. Due to these cellular colloidal chrystaloidal changes, infection takes place more readily:—That is to say, the infected area or infectious process is the result of cellular change instead of the infectious organism being the cause. Infection is probably of secondary entity. This may not be colloid or cellular metabolism, it may be apolarizing of ions.

One can continue discussion in this field for hours, but I shall conclude by saying that this case is one of primary cellular metabolism, probably due to glandular imbalance resulting in local endometrioses, which affected the gums and mucosa in the mouth, likewise in the intestinal tract (however I do not belittle the allergic angle). The local bacteriological findings are the result of secondary invasion on suitable soil.

PRESENT CONDITION—SEPTEMBER, 1945—

(*G.R.S.*)

It has now been several months since the patient has had any severe discomfort in her mouth. She states: "It is certainly better than it was and the gums do not smart any more." The color quality has changed, being less brilliant, and there is less of the white film. The patient has had but three prophylaxis treatments in 1945. The mouth was examined on August 27, 1945, and the improvement is worth noting, although not satisfactory. The palatal and lingual gingival tissues are still practically normal with the exception of the lower left bicuspid and molar area. The labial gingiva over the lower and upper left lateral, and the lower left bicuspid is nearly normal in color and appearance.

A recent bacteriological examination has been made and has again revealed the presence of *Monilia albicans*.

* Woodburne, A. R. *J. Periodont.* 15 (1): 7-17, Jan., 1944.

Nutrition and Dental Health

IV. VITAMIN A.

By DOROTHEA F. RADUSCH, D. D. S., B. A., M. S., *Minneapolis 2, Minnesota*

THE HISTORY of the substance called vitamin A started when Hopkins recognized in the year 1912, that animals could not live on a diet of pure protein, carbohydrate, fat and water, even when necessary inorganic materials were added. Shortly after, Osborne and Mendel found that cow's milk possessed a disease protecting substance which was soluble in fat. This substance, the first recognized vitamin, was later called vitamin A.

Recently, Booher (Wohl, *Dietotherapy*, W. B. Saunders Co., Philadelphia. 1945) summarized the accumulation of data on vitamin A. Her chapter was used as a reference for the first portion of this discussion.

Vitamin A, so far as is known, occurs exclusively in animal tissues. A pro-vitamin in the form of carotenoid pigments is present in certain fruits and vegetables which may be converted into vitamin A by the livers of animals. Thus part of the vitamin necessary to man is obtained from preformed vitamin A and part from pro-vitamin A.

Animal livers and kidneys contain practically all their vitamin A values as preformed vitamin A. The leafy green and yellow vegetables, yellow fruits, and red tomatoes are the chief articles in which the vitamin A is entirely in the form of pro-vitamin A. Egg yolk, and milk carry part in each form.

In a well-fed person, the liver gradually acquires an increased stock of vitamin A which normally reaches its peak only in adult life. Yet livers of adults show extraordinary variation in vitamin A content. This might be expected in view of the variations of the vitamin A values of common diets. What are the advantages of large liver stores? We do not know except that possibly they offer protection against vagaries in vitamin A intake and against possible shortage of vitamin A when food intake is low or needs are increased.

It is known that some adults can subsist for many months, even a year on a diet practically devoid of vitamin A value without showing recognizable adverse signs or symptoms of vitamin A deficiency.

Vitamin A is better absorbed in normal persons than carotene. From present evidence, it would appear that about 50 per cent of the vitamin A-active carotenoid pigments is absorbed. The efficiency of the absorption of both decreases in normal individuals as the size of the oral dose is increased. Therapeutically, therefore, it would seem better to administer vitamin A in several moderate doses a day than in a single daily massive dose.

POTENTIAL HARMFULNESS OF MINERAL OIL TO VITAMIN A UTILIZATION

It should be emphasized that many investigators have observed that the administration of mineral oil simultaneously with carotene or shortly after or before meals will prevent the absorption of carotene. To a smaller extent, this holds true for vitamin A. This is true both with plain petrolatum (mineral oil) and the petrolatum and agar mixtures.

The long established and common practice of recommending use of mineral oil instead of salad oils in reducing diets because of the lower calorie value is to

be condemned. Such practice is detrimental to the most effective use of the vitamin A value of the foods consumed.

This relationship between mineral oil and the potential vitamin A-value of foods eaten assumes greater significance because of other habits peculiar to these war years. Fats and oils have been grouped with meats in the red-point rationing program. Since many preferred to use their red points for purchase of meat, point-free mineral oil was often substituted for cooking oils not only as an ingredient of salad dressings, but for deep-fat frying of potatoes, doughnuts and croquettes. These are procedures which should be stopped, both by private and commercial users.

If petrolatum is to be used for relief of constipation, it should be taken just before retiring, never near or at meal times.

PRESERVATION AND STABILITY OF VITAMIN A IN FOODS

The carotenoids in foods are quite stable to ordinary methods and temperatures of cooking unless exposed on the outer surfaces of the food to direct flame or fried in fat heated to a high temperature. In such case, it is believed that some of the vitamin A value of the outer layer of the food may be destroyed. Fine mincing of leafy green vegetables causes substantial loss of vitamin A value as a result of oxidative processes catalyzed by leaf enzymes. Thus, they should not be prepared until shortly before use.

Whether cooking leafy green and yellow vegetables improves absorbability of their vitamin A value is not entirely clear, but a few experiments with adult human beings seem to indicate that it does. Further, from experiments, it would appear that the carotene in such foods as cooked spinach and cooked green peas is better utilized for that function of vitamin A which has to do with the maintenance of normal dark adaptation of the eye in adult human beings than carotene dissolved in vegetable oil (cotton seed oil). Fats and vegetable oils when combined with the carotene-carrying foods seem to improve absorption of carotene.

Frozen foods if properly blanched, retain their vitamin A values extremely well, while canning is not appreciably destructive of these values.

Fortified oleomargarine contains as much vitamin A on the average as does butter, each carrying approximately 199 I.U. per teaspoon. From both the vitamin and fat utilization, it may be safely used in place of butter.

PRINCIPAL FOOD SOURCES OF VITAMIN A

Principal food sources in the average American diet are leafy green and yellow vegetables, milk-fat and egg yolk, animal livers and yellow fruits. Compilations on food consumption data indicate that roughly 50 per cent of the daily 5000 International Unit standard is derived from preformed vitamin A (animal sources—1 pint milk, 1 egg, butter and cream) and 50 per cent from carotenoid pigments. Thus, without daily consumption of the highly colored green and yellow vegetables and yellow fruits, it is unlikely that the intake would be sufficient to meet the standard.

Increasing the family's vitamin A consumption is good for young and old, it appears from studies of rats reported by Dr. H. C. Sherman and Dr. H. L. Campbell, of Columbia University, to the National Academy of Sciences. Liberal intakes of this vitamin tend

to postpone aging and to increase length of life, Dr. Sherman and colleagues have previously reported. Now they find that the offspring in rat families on the liberal vitamin A intake grow somewhat more rapidly and with less individual variability. This indicates that liberal vitamin A has both a favorable and a stabilizing influence on growth. This favorable, stabilizing effect on rat growth was observed with vitamin A intakes two and four times higher than the intake considered fully enough to meet the rat's nutritional needs. —(*Science* 102:10, Supplement. July 27, 1945.)

One must always keep in mind that experimental deficiency studies have brought to light notable species differences in metabolic processes. Thus results in one species cannot be applied to another without verification. Nevertheless, animal experimentation is valuable as an indicator for possible human responses.

VITAMIN A AND CUTANEOUS LESIONS—INCLUDING ORAL LESIONS

Since laboratory research has shown the importance of a proper amount of vitamin A for the maintenance of health of certain epithelial tissues, the question is often asked as to its relationship to the oral epithelium in periodontal conditions. To properly discuss this question, one must have in mind the histology of the oral epithelium as well as the known action of vitamin A upon epithelial tissues.

Sullivan and Evans (*Arch. Dermat. Syph.* 51:17, 1945) reviewed and discussed the subject of vitamin A and cutaneous lesions fully. They said:

> The lesion of vitamin A deficiency has been defined by Wolbach and Howe (1925) as atrophy of many glands, arrest of growth, emaciation and replacement of many different single-layered epitheliums by stratified keratinizing epithelium. The process of the change from cuboidal or columnar type of epithelium to the stratified type is known as keratinizing metaplasia, and it has been demonstrated numerous times in various epithelium other than skin. There is no confusion regarding the primary histologic alterations and the resulting lesions of the eye, the paraocular glands and the respiratory, gastro-intestinal and genito-urinary systems.
>
> There has developed an acceptance of the concept that the cutaneous lesion due to vitamin A deficiency is similar to that of other epithelium and that it is the result of so-called keratinizing metaplasia.
>
> On histologic grounds, this concept is difficult to accept. In the skin the epithelium is of the stratified keratinizing type (normally). Therefore, keratinizing metaplasia in its literal meaning is an impossibility unless atrophy, which precedes the "metaplasia" is so profound that a single-layered epithelium is produced.

After careful review of all early experiments on vitamin A deficiency and comparison of results with other experimental research, Sullivan and Evans call attention to the fact that in the earlier experimental studies of vitamin A deficiency, the deficiencies which existed were actually complex and included deficiencies of B-complex, fat, and essential fatty acids as well as of vitamin A.

In more recent experiments, when young rats were fed a diet deficient only in vitamin A, according to schedules set up using the newer knowledge of the vitamin content of foods:

> There resulted a deficiency disease, the gross and microscopic signs of which differed from previously recorded descriptions of vitamin A deficiency. In such animals there were no gross cutaneous alterations as described by the earlier investigators, except during the very late stages of the deficiency when it is impossible to exclude the effects of inanition and superimposed deficiency.

Sullivan and Evans also showed that the signs of vitamin A deficiency in the

rat may be delayed by increasing the amount of vitamin B complex and the deficiency signs may be precipitated by inadequacy of vitamin B and fat in the vitamin A-deficient diet.

These observations indicate an interrelationship of vitamin A and the vitamin B complex and suggest the possibility that vitamin B complex deficiency preceded or accompanied vitamin A deficiency in man when follicular hyperkeratoses were produced as described by Frazier and Hu (*Arch. Int. Med.* 48:507, 1931).

From the data presented by Sullivan and Evans' thorough discussion and experiments, one may conclude that cuboidal or columnar epithelium may undergo keratinizing metaplasia due to vitamin A deficiency, but that tissues normally stratified and keratinized, such as skin and gingiva, cannot be so affected.

The epithelium of the gingiva is normally keratinized and many layered, according to Orban *(Oral Histology and Embryology,* 1944); although, he also makes the somewhat conflicting statement that,

All transitions from non-hornified to parakeratotic [partial or incomplete hornification in which cells have lost their boundaries is parakeratosis] and hornified epithelium of the gingiva have to be considered as within the range of normal (p. 268).

Schour and Massler have recently reviewed the data on vitamin A as it applies to oral structures (*J.A.D.A.* 32:714, June, 1945). They state that,

Abels, *et al* (*Cancer Res.* 2:381, June 1942), suggest a possible relationship between vitamin A deficiency and hyperkeratosis of the gingivae and oral leukoplakia.

Abels and his co-workers however, do not make such an inference in their published report. They used plasma levels of vitamin A and urinary excretion of riboflavin as criteria to determine whether or not their experimental patients suffered from a general avitaminosis. Such data were used to confirm their findings as to the incidence of simple dietary deficiencies as previously derived from dietary histories. No attempt was made to associate the plasma level of vitamin A with the oral lesion present. The only test supplement given was large quantities of Brewer's yeast for varying periods of from two to ten months to patients with papillary atrophy of the tongue or oral leukoplakia. Sixteen or seventeen patients with papillary atrophy of the tongue showed subjective and objective improvement, while fifteen of twenty-three with oral leukoplakia showed improvement. In four of the latter, there was marked improvement. The Brewer's yeast supplement provided large quantities of thiamine, riboflavin, nicotinic acid, pyridoxin, pantothenic acid, protein and sulfur amino acids.

Sullivan and Evans' conclusion that tissues of the stratified keratinizing type cannot undergo keratinizing metaplasia, such as is recognized as occurring in single layered epithelium as a result of vitamin A deficiency, is of special interest in view of research findings on the relationship between periodontal tissue status and vitamin A intakes. No recognizable clinical or histo-pathologic changes were found in the periodontal tissues of monkeys fed a vitamin A deficient (partial deficiency) diet (Topping and Fraser, *Public Health Report* 54:416, 1939; Tomlinson, *Ibid:* 431). Further, although according to their dietary histories, intake of vitamin A was very low in adult human beings, such intake was not significant statistically when correlated with periodontal condition (Radusch, *J. D. Res.* 18:305, 1939).

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Studies in Dental Nomenclature: A Series

III. PERIODONTAL NOMENCLATURE *(Continued)*

By HARRY LYONS, *Richmond 19, Virginia*

IN THE first article of this series, several undesirable language trends in dental nomenclature were discussed. The second article of the series dealt with the nomenclature of the more commonly occurring periodontal diseases.

Probably the least explored aspects of periodontology are the neoplastic diseases of the periodontium. This may be due to a number of factors. The relative infrequency of the neoplastic disease as compared to the frequency of the inflammatory and degenerative diseases in this field and the comparatively limited knowledge of neoplasms in general may explain the situation. However, the incidence of neoplastic disorders of the periodontium appears to be increasing, due in part to new etiologic agents such as sodium dilantin. This and more careful diagnostic studies are bringing a greater number of cases to light.

The prevailing nomenclature generally used in this area of periodontology is altogether inadequate and confused. Much of the confusion in this sphere arises from the arbitrary use of one term: *epulis*. The word *epulis* is of Greek origin, having been derived from *epi* (upon) and *oula* (gums). The Greek word *epoulis*, meaning "gumboil," has the same etymologic derivation. From this it would appear that any lesion, neoplastic or otherwise, may be termed an *epulis*. However, authors have arbitrarily reserved the term for certain tumors occurring "on the gums."

The types of tumors to which the term *epulis* is applied vary widely among authors. Some use the term only in reference to the peripheral giant cell tumors arising from the periodontium; others use it in connection with gingival granulomas and fibromas; still others use it to designate periosteal tumors on the alveolar process without regard to their histologic structure. There is no etymologic basis for any limitation in the use of the term. Its only logical use is to designate the locale of a lesion. In view of this limited usefulness and the prevailing confusion regarding its definition, it would appear desirable to discard the term *epulis* altogether. The term certainly has no place in accurate diagnostic studies. Rather should periodontal hypertrophies, hyperplasias and true neoplasms be designated on the basis of their tissue characteristics. In instances of known causation, etiologic designations may be added to the names of such lesions.

The hypertrophies and hyperplasias of the gingivæ are varied in their causation and character. The true neoplasms which occur in the periodontal field include a wide variety of tumors of both benign and malignant types. It is not within the purview of this discussion to enumerate and describe these. The point emphasized is that accurate diagnostic studies, good teaching, and the scientific literature covering this sphere call for terms that are informative and accurately descriptive in themselves. These lesions, designated by fully informative names based on tissue structure and possibly causation, may then be *located* in this field by the use of such adjectives as *alveolar, gingival*, or

(Continued at foot of facing page)

In Our Opinion

PERIODONTIA PROCEDURE

Initial Statement

HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

THE local treatment for periodontoclasia and gingivitis is to remove the irritants and restore the natural tone of the tissue by massage. Calculus and other root surface roughness are removed by careful root surface curettage. Food impaction and traumatic occlusion are corrected by restorations, replacements, and conservative reshaping of interfering cusps and outstanding points. Systemic causes of tissue alteration and lowered resistance are diagnosed and treated wherever possible in coöperation with a physician. The patient is carefully instructed in methods of home care by which the tooth surfaces are kept clean and the congested chronically inflamed gingival and periodontal tissues are massaged until congestion and inflammation are eliminated and a healthy periodontium is established. When this result is satisfactorily accomplished, the soft tissues will hug the tooth so tightly as to eliminate space between tooth and gingiva. In some cases the epithelium lining the pocket apparently reattaches to the root surface sufficiently to reduce greatly the clinical pocket depth. This disease process will be stopped and with reasonable care the teeth can be saved indefinitely in health.

The steps in the procedure to treat a case of periodontoclasia are as follows:

1. A complete set of dental radiographs (fourteen regular films and four posterior bite wing films).
2. Accurately made record study casts.
3. A complete dental examination and diagnosis. Systemic therapy or diagnosis if indicated, is arranged.
4. Education of the patient regarding his dental condition and the treatment recommended.
5. Extraction of teeth which are evidently beyond hope of restoring to health and function.
6. Instruction of the patient in home care technics. (To be repeated at subsequent visits until satisfactory.) Practitioners generally prefer to postpone this step until root surface treatment is near completion to avoid the tightening of the gingiva which home care produces.

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periodontal. These principles lead to such informative and descriptive designations as gingival carcinoma, gingival fibromatosis, hormonal gingival hyperplasia, traumatic gingival granuloma, and alveolar osteoma. Only by the use of such nomenclature principles can exact information be recorded and transmitted.

(To be continued)

7. A preliminary cleansing in which salivary calculus, soft deposits, and stains are removed from the exposed tooth surfaces. (This step is often omitted by practitioners who treat each tooth completely on both supragingival and subgingival surfaces.)
8. Subgingival root surface treatment of as many teeth as can be completed at one sitting. This is continued at each sitting until all root surfaces are smooth and free from bacterial slime, calculus, and other roughnesses.
9. Final polish of the exposed surfaces of the teeth.
10. Final inspection of root surfaces and gingival tissues.
11. Surgical resection of overlying gingiva where periodontoclasia has progressed so deeply that surface massage pressure will not reach the deepest zone of congestion.
12. Adjustment by reshaping of interfering cusps and points causing traumatic occlusion.
13. Operative and prosthetic procedures to eliminate occlusal causes of periodontal disease. This involves restoring contact, contour, vertical dimension, replacement of missing teeth, and support for teeth which are tilted, drifted, or have weak alveolar attachment. Orthodontic treatment for drifted and malposed teeth is not considered practical for adult periodontoclasia cases by most American dentists but is advocated by many European authors.

Thorough systematic case history recording, oral examination, diagnosis, and treatment planning are essential to periodontia practice. Every condition and defect which might lead to periodontal destruction or loss of a valuable tooth must be discovered, recognized, and appropriate treatment planned to correct or offset it. A time should be arranged at which the health, nutrition, and dental history are recorded and the mouth and teeth examined with the aid of an X-ray film set, study casts, disclosing solution, pulp tester, and other essential paraphernalia. The dental examination should cover in detail the condition of oral mucosa and tongue, mouth care, oral hygiene, condition of gingiva and periodontal structures, occlusion as a whole and of individual teeth, replacements, individual teeth as to cavities, erosion, and abrasion, restorations, pulp condition, root canal fillings, periapical pathosis, etc. Defects related to the function of the teeth need particularly detailed attention as it is these defects which cause failures in many cases where root surface instrumentation and home care are ideal but oral diagnosis is weak. Such defects include lack of function, mobility, missing, impacted, tilted, rotated, extruded, and otherwise abnormally placed teeth, signs of occlusal trauma, faulty contacts, mouth habits which bring abnormal strains on the teeth, malrelations between jaws, flat occlusal surfaces, interfering cusps, and others. As defects are noted, the appropriate treatment may be planned, or treatment planning may be postponed until the examination is complete. Conditions which cannot be determined definitely at the time of the examination should be noted and treatment planned depending on the determination of the condition. Contingencies; that is, untoward developments if treatment is neglected or during

the course of treatment or after treatment is completed, should be thought of and noted to be discussed with the patient.

The education of the patient as to dental defects discovered, the contingencies and the plan of treatment in his case is a most valuable step which is too often neglected. A good piece of education at this point creates an understanding and a coöperative patient and an enthusiastic friend of dentistry. In the busy practice, especially if the dentist is not gifted as an expositor, much of this educational work may be delegated to a well-trained secretary. In many instances such a woman can give the patient a clearer understanding than the dentist can. In no case, however, should this step of patient education be neglected, for neglect at this point is the cause of nearly all the difficult and disagreeable misunderstandings and controversies between the dentist and his patients.

The general order of procedure in instrumentation is first to cleanse the exposed surfaces of the teeth of all salivary calculus, tobacco smoke tar, superficial stains, and bacterial deposits. This is all done with steel instruments. The teeth should be clean to the gingival margins after this treatment. It is seldom necessary to use abrasive agents like pumice for this purpose and much harm may be done if they are used. Most of the superficial discolorations are due to deposits on the tooth surface which do not come off readily by polishing unless a coarse abrasive like pumice is used. Pumice used with brush wheels or rubber polishing cups inevitably scours away surface enamel adjacent to the deposits. It is almost impossible to use brush wheels without abrading the gingiva and initiating a series of very sore spots. With skill and experience it takes only a few minutes in most mouths to remove the deposits with steel scalers and this is so much more accurate than indiscriminate scouring with abrasive powder that it is the method of choice.

After the exposed tooth surfaces are clean, the subgingival root surfaces can be approached working from clean tooth crowns to coated roots. Each root should be completed before going to the next. Or similar surfaces on two or three teeth may be treated at a time to save change of instrument position. Only as many roots should be touched as can be completed in one sitting. The teeth should be taken in regular order so that none may be missed. What the order is, is unimportant provided the operator habituates himself to using the same order in all cases. If he skips around, he is almost certain to leave some tooth untreated at some time. All teeth should be treated whether or not pockets were noted at the time of the examination. If no pockets or hard subgingival deposits exist, it takes only a moment to go over the subgingival surfaces. In any event the subgingival surfaces should be curetted lightly to remove bacterial slime on the tooth surface. If this procedure is not followed, much of the value of the treatment will be missed. The soft tissues should not be curetted, punched, or injured any more than is necessary to reach and curette perfectly all detached root surfaces with well adapted instruments. Only sufficient root surface curettment should be done to free the surface of foreign deposits and of roughnesses. Cementum shaving in excess of this reduces the thickness of cementum and will in time lead to sensitive spots of exposed dentine.

Overhanging filling margins, ledges of enamel at the cervix, rough enamel and cementum should be reduced to smooth rounded contours as much as possible. Every roughness is not only irritating mechanically to the overlying gingiva, but also acts as a crevice for bacteria-fungus accumulation.

After the root surface treatment is completed, the exposed surfaces of the teeth are polished with wood points in a porte polisher using a paste of flour of silex (triple x) or other fine polishing agent and water or glycerine. At the same time the gingivæ are massaged by compressing the gingival margin with the wood point at each polishing stroke. When all accessible axial tooth surfaces are polished with the wood points, the proximal surfaces are polished with dental floss in which the fine abrasive paste is incorporated by passing the floss through the paste. The floss is used in the same manner as advocated for the patient in the home care procedure. The occlusal surfaces seldom need polishing except in non-occluded teeth as they are well scoured by food during mastication. A dry cloth over the finger end, dipped in dry silex can be used to polish the surfaces accessible to the finger. This gives a polished feeling to the teeth which is very pleasant to the patient.

When the polishing is completed, the gingival tissues should be examined in detail under a strong light for dark spots which may indicate possible scales of calculus still remaining, and for soft exuding areas which indicate inadequate treatment by operator or patient. If the final polish is given before healing of treated teeth has had time to take place, an appointment should be made for a later inspection. No case should be considered completed until all exudation and signs of congestion and inflammation have disappeared and the tissue overlying pockets has become pink, firm, resistant, and the sulcus is free of soreness or bleeding upon probing.

The patient should be put on a recall list and recalled for treatment at sufficiently frequent intervals so that recurring inflammation may not go to the point of causing further periodontal destruction. Most patients will not be able to maintain the teeth and gingivæ in such perfect cleanliness and health but that slight deposits will form here and there, both on the exposed tooth surfaces and beneath the gingival margins. It is these deposits that must be removed by treatments at appropriate intervals. At these appointments the patient's home care is also rechecked. Treatments at intervals of six months are sufficient to maintain the periodontal structures of most patients in health if home care is good. Until home care is well established more frequent treatments may be indicated.

In addition to the periodontal treatment every six months, every patient should be recalled once a year for dental examination and diagnosis. This includes a new eighteen film set of radiographs, but not new study casts unless changes in tooth position or level of gingiva make additional cast records desirable. The examination including case history should be just as thorough and systematized each year as at the first appearance. Each time that an examination is made the patient should have time arranged for discussion and education regarding defects discovered, possible contingencies, and treatment recommended.

With this sort of routine there is very little chance that any defect will develop to the point of loss of a tooth before it is discovered and treated. Loss of a valuable tooth in a patient who desires regular dental care is with few exceptions an indication of neglect by patient or dentist and usually it is the dentist who is to blame. No one is one hundred per cent infallible, but it should certainly be a rare exception that a patient having regular care, whose teeth were not so hopelessly involved at the first visit, but that they could be restored to health, should have to lose teeth during the course of his remaining lifetime.

By systematic administrative routine in the office none of the details of procedure as outlined need involve undue expense or time. An intelligent capable dental secretary-assistant can easily be taught to take and develop the dental radiographs. She can also make up the study casts after the dentist has taken the impressions. Impression taking seldom needs to take more than fifteen minutes of the dentist's time. The dental secretary can obtain by questioning most of the data needed for health and dental history. If dietary study is indicated, she can be taught to compute the composition of the average daily diet from a week's diet as submitted by the patient. She can take the dictation and help in the dental examination by studying the radiograms as the dentist explores with instruments the teeth and gingival pockets. An examination with such help, including notes on contingencies and a complete plan of treatment will usually require from thirty to forty-five minutes. The secretary then takes the dictated notes and makes up a typewritten record for the file and later goes over this material in the education of the patient. The total time the dentist needs to spend prior to starting treatment should seldom be over one hour.

Excepting in a large group practice where the time of a hygienist can be kept occupied in cleansing and polishing the exposed surfaces of the teeth and instructing patients in home care technic, a dental hygienist is not of great economic value to a periodontist. The time she can save in the treatment of a new periodontia case is approximately one hour out of four or five on the average. If the periodontist also does the restorative and replacement dentistry, the saving is far less proportionately. He can not even delegate her to give the twice a year check up treatments without himself seeing the patient to be sure that pockets have not opened again and that subgingival deposits are all removed. Combining the duties of secretary-assistant and dental hygienist is not practical unless there are hours during the week when the hygienist can work on patients uninterrupted by her duties as assistant and secretary.

It is not enough for the periodontist to be a skillful technician in root surface treatment and teacher of home care to the patient. The successful treatment of periodontal disease and maintenance of the teeth and oral structures in full function, comfort, health, and beauty throughout the lifetime is the function of every dentist and especially of the periodontist. This requires establishment of a routine procedure embracing dental office administration including a recall system, case history taking, dental examination, diagnosis, treatment planning which is complete and comprehensive, education of the

patient in dentistry as related to his dental condition, and teaching the technics of home care, in addition to competent technical treatments according to the plan.

Whether or not the periodontist does the dental operative, prosthetic, and orthodontic procedures which may be necessary fully to restore and maintain the mouth in health, function, comfort, and beauty, I am convinced that he must control these procedures. Otherwise there is divided responsibility. If the prosthetic procedures do not fully support the periodontal requirements, failures result. Teeth with supporting tissues weakened by periodontoclasia need watchful care and comprehensive controlled procedures in order that hygiene and mechanical equilibrium may be maintained. The periodontist should be in position to direct the care of such teeth.

Discussion

LAWRENCE A. DONAHOE, D.D.S.,
Omaha 2, Nebraska

I believe that the "Initial Statement" covers the ground quite thoroughly.

If there is any reason to believe that a systemic condition due to some of the blood diseases such as syphilis, or especially leukemia, or to some of the kidney conditions, is contributing to the cause in a case of periodontoclasia it is advisable to always have the coöperation of the patient's family physician or a diagnostician.

In the statement of steps and procedure, I feel that Step No. 5 should be a good physical examination as the health and the age of the patient has much to do with the prognosis and the end result of the case.

As to Step No. 13, referring to the orthodontic treatment of drifted and malposed teeth, I agree that orthodontic treatment is not considered practical in the adult periodontoclasia cases. I have had orthodontists refer cases to me for treatment, the patient having been given to understand that they were to return later for further orthodontic treatment after my work was completed. In all of these cases I endeavored to discourage the orthodontist and the patient from having anything done as it would only disturb the condition of the healing attachments and would be impractical.

I agree with the idea that a special appointment should be arranged in which the health, nutrition, and dental history are recorded, and the mouth and the teeth examined with the aid of an X-ray film set, study casts, disclosing solution, pulp tester, and all other essential paraphernalia; also the health condition of the oral mucosa, tongue, mouth care and oral hygiene, periodontal structure, occlusion as a whole, replacements, cavity restorations, pulp conditions, root canal fillings, and periapical pathosis. This and the following paragraph are very essential and often neglected in part or whole by a good many operators.

In the education of the patient as to the condition of oral and dental defects, I feel that the dentist is the only person qualified to instruct the patient and not a well-trained secretary or office assistant. The first impressions are the most lasting ones and the dentist should educate himself in the proper manner of instructing and educating his patients. This would make less likely, disagreeable misunderstandings and controversies between the dentist and the patient.

It is stated under the head of general procedure in instrumentation, that it is seldom necessary to use an abrasive agent like pumice. On the contrary, I feel that it is necessary to use the flower of pumice mixed with glycerine to remove some stains and plaques, applied with a rubber polishing cup. It is true that brush wheels do abrade the gingiva and the improper use and carelessness in the application of rubber cups will also destroy and scour away some of the enamel surfaces of the teeth. I use several applications of the disclosing solution suggested by Sorrin and Miller in their Text-

Book, and find this solution a great help in the removal of stains and plaques. It helps also to break down some of the calculus deposits. If there is any abrasion or injury to the gingiva, we suggest that the gum tissue be made as dry as possible and an application of tincture of benzoin be used to relieve this condition.

As to the procedure of the cleansing of the root surfaces in a regular order I feel that the method suggested is splendid and very thorough.

BERNARD D. FRIEDMAN, D.D.S.,
Chicago 2, Illinois

I would like to go on record as agreeing wholeheartedly with the recommended treatment, in the "Initial Statement." The only points for discussion relate to procedures.

Dr. Leonard states that the bacteria concerned in periodontal conditions are the ordinary bacteria and fungi of the mouth. This is indeed true and should be a deterrent to the use of so-called bactericidal drugs which are offered to the profession.

When a mouth presents with apparent gingival infection and impaired circulation in the gingivæ, the first procedure should be to cleanse the mouth of all the dead bacteria, fungi, desquamations and local irritants.

In doing this thoroughly much of the later subgingival curettage can be lessened and made easier.

The mouth care which is intended to maintain oral health after treatments should be instituted at the *first visit*.

At the *first* visit the whole dentition should be gone over as carefully as possible with proper instruments to remove as many of the local irritants as possible.

It is not necessary to plane and polish the tooth surfaces one or two or three teeth at a time.

Then the gingivæ should be brushed thoroughly, by the operator, with a toothbrush and a mild, powdered dentifrice. The patient should be given a hand mirror to watch the procedure so that he may learn *at once* the proper hygiene of the gingival tissues.

In doing this at the first sitting and instructing the patient to do it similarly at home twice daily, it will be found that the major engorgement of the gingival tissues is greatly reduced and the root surfaces are then more easily accessible and the curettage is less painful, less difficult, and causes much less bleeding.

I would like to emphasize that the most important duty we have to perform for these patients, as in any bodily ailment, is the determination of the cause or causes of the abnormal conditions. Hence, after the mouth is cleansed, the diagnosis must be thorough and intelligent, and the contributing factors in the disease must be eliminated, i.e. all local irritants must be removed; inlays, fillings, and crowns rendered nonirritating at the gingival margin; the occlusion rendered innocuous i.e. the bite should be balanced, or opened; missing teeth should be replaced with fixed restorations wherever possible, traumatic occlusion should be adjusted; then, and then only, will the periodontoclasia be arrested and the mouth remain in continuing health.

SIDNEY S. FRIEDMAN, D.D.S.,
Memphis 3, Tennessee

Dr. Leonard's steps in clinical procedure are excellent and form a basis of sound periodontal practice. Some discussion of the etiology of the series of slight chronic ulcerations or inflammations mentioned would be helpful.

Diagnostic routine is emphasized. The points mentioned in the steps Nos. 1, 2, and 3 are a prerequisite of periodontal treatment.

Step No. 4 brings out a good point in practice management and must be varied according to the appreciation of the patient receiving the service. Also it should be impressed upon the patient that he must accept his full share of liability in the success or failure of periodontal treatment. The patient must be taught to practice rigid personal mouth hygiene.

Step No. 5, "Extraction of teeth which are evidently beyond hope of restoring to health and function"—it would be well to bring this highly important point of diagnosis into sharper focus. I have found that when, radiographically, a tooth has one-half or less of its remaining bone structure, there is a questionable prognosis. This is more certainly true if the architecture of the bone shows wide spaced trabeculation. Molars with an open involvement of the

bifurcation or trifurcation present a hopeless prognosis. Clinically where a tooth has a range of movement both lateral and apical, indicating that there has been extensive destruction of the principal periodontal fibres, there is little hope offered in treatment. Some system of classifying terminal pathology, indicating teeth that can be treated and those in which treatment is contra-indicated would be valuable in a discussion of this type. Frequently, one is impressed with the fact that the criteria of tooth preservation is not so much pathologic as geographic.

Steps No. 6 through No. 13 present an orderly approach to rational treatment. While the sequence might be varied to meet the immediate demands of the specific case Dr. Leonard's procedure is thorough and consistent.

I must disagree with the statement "it should certainly be a rare exception that a patient having regular care, whose teeth were not so hopelessly involved at the first visit, but that they could be restored to health, should have to lose teeth during the course of his remaining lifetime." "Remaining lifetime" is a variable factor and it is to be hoped that many of our patients will outlive the teeth we aid in maintaining in a satisfactory state of health for a sufficient period of time to more than justify the treatment. Also teeth are lost because of other pathologic processes than periodontal involvement.

Concerning the use of a dental hygienist the periodontist must himself see the patient at check-up visits. Unfortunately, it must be admitted that lesions do develop in the mouths of patients receiving periodic prophylaxis not only by a dental hygienist but by a dentist as well. A dental hygienist is most helpful in my practice and I consider the time saved worthwhile in preparing the patient for me and in subsequent prophylaxis.

Periodontists treat a type of pathology that is not immediately dramatic. Recognition of deviations from normal are important as the success of all treatment is in direct ratio to early diagnosis.

EDWARD L. GAMBILL, D.D.S.,
Lexington 3, Kentucky

I heartily commend Doctor Leonard for a very excellent "Initial Statement." He shows a wonderful spirit in asking for the brickbats of honest opinion. He believes that the "consciousness of error is the beginning of wisdom." A true scientist, which every dentist should be at heart, holds to any opinion, procedure, or technique only until better ones can be found.

The condition of the mouths of the American people is a disgrace to our profession and to our nation. Periodontoclasia through its ramifications is one of our greatest killers. Too often, far too often, the disease is not recognized. Many dentists, physicians, and nurses are filling premature graves because of our failure to recognize and treat periodontoclasia effectually. Many other diseases, such as tuberculosis, infantile paralysis, cancer, typhoid, and the various contagious diseases have been publicized and combatted, but there has been no impressive educational effort to combat periodontoclasia. This educational work should be done, to a large extent, by non-dental organizations, the individual dentist lending any moral or financial support to it that he may consider desirable, but holding no office in such an organization. The public should be made periodontia-minded, and if it is so made the dental profession will need to become more periodontia-minded, for reasons of self-preservation. The periodontists of this country should show the way.

As to the etiology of periodontoclasia, it has been well stated by Doctor Leonard. However, I believe more and more emphasis must be placed on inefficient hygiene as the most outstanding causative factor. The food line of today is the gingivitis of tomorrow. The gingivitis (pre-periodontoclasia) plus traumatic strain in many cases is the periodontal pocket of a few months or a few years hence. Periodontoclasia is a disease that should be "treated" before it develops. Microörganisms do not thrive where they get no food. They like a sulcus or pocket, and for this reason I believe in the elimination of the pocket without too much curetting. I do curetting in a general way, but not two or three teeth at a time as formerly. Curetting pockets or doing surgery in the mouth, whether it be extractions or resecting of gum tissue, without effective premedication is for the most part, not using surgical methods. The factor of microörganisms should be eliminated to a very

great extent prior to such procedures. I do not get much regeneration of the pericemental fibres except occasionally in favorable cases where the pocket is confined to only one surface. Resecting of the pocket to the approximate depth of the sulcus with proper regard for appearance is the easiest and most practical way of pocket elimination. If this is done and efficient mouth hygiene is immediately instituted and sustained, the results are invariably most gratifying. The system of home care which I am teaching my patients is something of a departure from the conventional ways, but it is giving astounding results. It has led me to the conclusion that Doctor Miller was almost right, that periodontoclasia is for the most part a filth disease.

I recommend the following ideas as good periodontology:

1. That dental floss become outmoded in favor of dental tape. Many dental tapes made at the present time are too small. This may be compensated for by doubling it and even quadrupling it when necessary.

2. Standardization of brushes as to size, shape and quality of bristles. We should tell the manufacturers what to make and our patients what to buy.

3. A national publicized crusade against periodontal diseases.

4. Emphasis on the importance of early diagnosis and a sense of co-responsibility on the part of the patient in the prevention and correction of periodontal lesions. Most cases are not referred to specialists until too far advanced.

5. Greater emphasis on effective hygiene as the most important basis of cure. The average toothbrush user gets about twenty-five per cent efficiency. About fifteen per cent of the people cannot or will not learn effective mouth hygiene.

6. Outlawing the cantilever one-abutment bridge as unscientific.

7. Discourage the greatly overused and abused gold shell crown.

8. Outlawing the long-spanned stationary bridge in the mouth and especially in replacing posterior teeth as being biologically, hygienically, and scientifically wrong, and recognizing the tissue-bearing or the semi-tissue-bearing removable types as more suitable.

9. Premedication as a prelude to surgical interference in the mouth.

10. Discouragement of limitation of practice exclusively to prophylaxis and periodontal surgery, by those who would specialize in this field.

11. The preservation of the third molars in all possible cases to prevent the weakening of the contactual relationship of teeth.

12. Recognizing and emphasizing improved hygiene as a most satisfactory solution of the caries problem.

13. Basing the periodic check-up on the patient's individual condition and the efficiency of his coöperation, rather than upon the customary six months idea. Some patients should be seen once a month.

I agree one hundred per cent with Doctor Leonard's last paragraph, in which he emphasizes the importance of clinical dentistry supporting the periodontia effort. Without the maintenance of this ideal our efforts as periodontists will largely fail.

W. M. GARDNER, D.D.S.,
Cleveland 14, Ohio

In my opinion the procedures set forth by Dr. Leonard are sound and productive of good results. This is particularly true in the simplex type of case. No doubt details of the procedures are modified by individual operators, but the essentials as outlined must be strictly adhered to in order to reach the desired goal.

I am heartily in accord with the emphasis placed on education of the patient. The psychology of teaching proper mouth hygiene is a subject that has been much neglected. There is very little in our dental curriculum that develops this side of the student. When his early and untutored attempts at teaching mouth hygiene are unsuccessful, they are apt to be discontinued. In a questionnaire submitted to a thousand patients, less than ten per cent had received instruction in mouth care from a dentist and fully half of these were instructed in ineffectual methods.

The use of study casts in the teaching of proper mouth hygiene should not be overlooked. It individualizes the problem and helps to stress certain points peculiar to the case in hand. With the newer materials impressions are quickly, accurately, and com-

fortably made, and time so spent will be well compensated for.

While the procedure of taking a new eighteen film set of radiographs each year may be an ideal one, it would hardly seem an economically sound or a necessary procedure. After the initial full-mouth study, judicious use of interproximal radiographs in most cases, I feel, would be an adequate check-up.

RAYMOND E. JOHNSON, D.D.S.,
St. Paul 2, Minnesota

If we are to succeed in the treatment of periodontoclasia, a definite procedure as outlined by Dr. Leonard must be followed in the handling of every case. The reason the dentist is successful in the making of dentures, porcelain jacket crowns, and other mechanical procedures is because he follows a very definite routine in his technic in all cases. As periodontists, we see many cases of advanced periodontoclasia where treatment has failed, because of an incorrect diagnosis and no plan of treatment.

Dr. Leonard has very thoroughly discussed what should be accomplished in each step of his outline of procedure. In my opinion, the order in his steps of procedure should be changed. Step No. 7 (a preliminary cleansing in which salivary calculus, soft deposits and stains are removed from the exposed tooth surfaces) I would prefer to make No. 2. More accurate study casts can be made if all calculus has been removed from the crown surfaces and certainly step No. 3 (a complete dental examination and diagnosis) when you check the conditions of restorations, individual teeth for cavities, open contacts, etc., should follow the preliminary cleansing. As to step No. 5 (extractions of teeth which cannot be restored to health and function)—I believe there is less chance of an undesirable reaction where the field has been made as clean as possible. The fact that the mouth has been made clean before the examination has a very good psychological effect on the patient as they are impressed by thoroughness.

JOHN OPPIE MCCALL, D.D.S.,
New York 21, N. Y.

I find myself in agreement with the statement on "Periodontia Procedure" in most respects. Considering space limitations it is a comprehensive statement of accepted theory and treatment. Many of the statements suffer from extreme condensation and I will take no exceptions to some of them for that reason.

I do find one statement regarding the course of the disease that tells only part of the story to which it relates; sequential to that, there is a recommendation as to the order of procedure in treatment with which I disagree.

Leonard states that following long continued and extensive periodontal inflammation the tooth extrudes from its socket causing a traumatic occlusion. That such events happen and in the order stated is true. But there are innumerable cases in which traumatic occlusion occurs because of an original malposition of the teeth and this in itself may become an original source of periodontal damage leading more or less directly to inflammation.

If the latter sequence occurs it follows that correction or relief of the traumatic occlusion should be done early in the course of treatment and not postponed till near the end as advocated.

I will be interested to see other comments on the points I have raised.

CARLOS H. SCHOTT
Cincinnati 2, Ohio

Our professional tomorrow depends greatly on our conception and promotion of the important factors, which bring about and maintain better dental health, and since the practice of periodontia is one, if not the most fundamental phase in dentistry, Dr. Leonard's very intelligent and understandable presentation on the subject of "Periodontia Procedure" is most timely.

In these post war days, when the many thousands of dentists will be returning to civilian practice with a high percentage of them having expressed a desire for postgraduate or refresher courses, it is imperative that there be a clear understanding of the etiology of periodontoclasia. Also, the teaching in these courses of proper and skillful periodontia routine should be adequate, in order to serve greater numbers, who so sorely need treatment. There are too few specialists in periodontia either full time or limited time practitioners, to take care of

the needs. Every dentist must be taught the importance of maintaining better periodontal health if we are to better serve dental patients. Dentists must be made to appreciate the value of proper mouth hygiene and to recognize the mechanical inharmonies, which lead to a breakdown of periodontal structures.

While a capable and intelligently trained assistant or secretary can be of great value in the educational instruction of the patient, I disagree on the point that a dental hygienist is not of great economic value to the periodontist (especially the part-time periodontist of which these are the greatest number), for she can do much of the preliminary prophylactic as well as the follow-up treatment, recordings, and educational work, if properly directed and limited to the work for which she is especially trained. All associates in the office should be trained to give this advice. She can be trained to give the twice a year or more frequent check-ups and recording of areas for our attention with a great economic saving of time, but I agree that her work should not be interrupted by having to do the work of secretary-assistant for many obvious reasons. To discuss a paper in which I am in almost complete agreement does not permit of anything less than a most hearty affirmation. If the full or part time periodontist wishes to contribute most to the progress of dentistry and to the better general health, he will try unselfishly to see to it that every dentist becomes at least a part time periodontist. And by decreasing our dental ills, there will be less demand for socialized dentistry.

The technique advised by Leonard is so complete, I see no need for additions.

ROWE SMITH, D.D.S.,
Texarkana, Ark.-Texas

Dr. Leonard has covered very completely in his paper the field indicated by its title, and I can find little to seriously question or with which to disagree. However, I do think the term "periodontoclasia" cannot be employed to cover all types of periodontal disease, and that it is inadequate and misleading terminology.

In most cases, sixteen apical views are required to furnish all necessary roentgenographic information, and two bite wings usually are sufficient to cover the interdental areas.

Intraoral kodachrome photographs should also be made at the time the roentgenographic examination is made, as these photographs will portray a fairly accurate clinical picture of the teeth and their supporting tissues before any treatment is instituted, which the roentgenographs, study models, records, etc., may not record as accurately. In addition, these photographs will be helpful in disclosing abnormal conditions which might possibly be overlooked. They will certainly help the patient and dentist to properly evaluate the result of the periodontal services when the case is completed.

Too often the so-called "old patient" fails to receive the same thorough and careful examination and attention accorded a new patient, consequently, potentially serious dental and periodontal conditions may be overlooked.

I heartily agree that periodontal treatment cannot be a separate and distinct phase of dentistry except in a few instances. Usually it requires a coördination of several or all of the phases of dentistry to restore the dentition and its supporting tissues to as nearly normal function as possible.

I see no necessity for using dental floss in home care if stimudents or round tooth picks are properly used in conjunction with good toothbrush technique.

Except in cases of true periodontosis or diffuse atrophy, I believe that loss of a valuable tooth by a patient who has had good periodontal treatment regularly, indicates that the dentist is to blame.

It is pleasing to note that the author stresses the importance of utilizing well-trained dental nurses and secretaries in order to render a better service to more patients. I certainly agree with the statements made in regard to utilization of hygienists, and their limitations.

In addition, I think the periodontist should have a good understanding of dental medicine and should be able to work sympathetically with the medical internist.

Even though it be generally considered that the author has covered periodontal procedure very thoroughly, I sincerely regret that he has failed to recognize, or at least to mention, the value of oxygen as a periontal therapeutic aid. Many of us who

have labored in the field of periodontia for years recognize that oxygen is the only drug or agent which will produce a favorable change in the character of the gingivæ and contiguous tissues. It acts within as well as on the tissues, whereas all other medicaments act only on the tissues. Oxygen therapy employed in conjunction with the treatment outlined in the paper under discussion will reduce the treatment time through rapid rehabilitation of the supporting tissues involved, and will materially help to prevent recurrence. It is a specific in the treatment of Vincent's infection in all its various forms.

I think it would have been well had more definite information been given in the "Initial Statement" as to how to determine whether the expected repair of the periodontal tissues has taken place following application of the various techniques of treatment, according to indications. It appears that Leonard has dealt vaguely with the matter of evaluating the results achieved, after sufficient time has elapsed for adequate healing and repair to take place.

I know that he is right when he emphasizes the importance of a complete oral and general systemic diagnosis and a thorough understanding by the patient of the nature of periodontal disease and his responsibility in establishing and maintaining periodontal and general health. The necessity for elimination of all diseased periodontal tissue and pathologic pockets and their accompanying irritating and septic matter is recognized. He has also forcibly stressed the importance of the restoration of the dentition to as near normal function as possible, the correction of any nutritional deficiencies and other general health disturbances, and the institution and maintenance of oral hygiene and periodontal stimulation.

Periodontal experience and training will prove that the statements contained in the last two paragraphs of the paper are fundamentally true and must prevail if the best interests of the patient and periodontist are to be served.

I would like to suggest recognition of a common condition whereby the lower anterior teeth are displaced very extensively, causing a buckling condition. Special treatment is required to control the periodontal disease usually associated with this condition.

Concluding Statement

(Prepared by the Editor at the request of Harold J. Leonard, Director of the "In Our Opinion" Department.)

IN MOST mechanical procedures in dentistry there is a generally accepted order of steps because the various ones are so dependent upon each other. It is not possible to perform Step No. 2 before Step No. 1, but after Step No. 1 is taken, the others follow in regular succession. In periodontia, however, it is quite practical to reverse the order to some extent and for this reason much confusion often results. From the preceding "Initial Statement" and "Discussion" one notes a difference of opinion as to the order of procedure.

For example, Drs. Gambill and Johnson regard as important the postponement of extractions until the field is properly prepared for the operation. From the standpoint of surgery this is correct. However, there are conditions where the extraction of certain teeth may be urgent. If the patient's health requires prompt removal of infection, or if a condemned tooth is very loose and annoying the patient, or for any reason is causing pain which cannot otherwise be relieved, extraction is indicated at once. Where the risk in extraction is less than the advantage to be gained, one might take a chance. However, to make a practice of removing all condemned teeth from a mouth filled with infectious material does not seem to indicate sound judgment. It is commonly done, but even that does not justify it. The field should be properly prepared for any surgical operation.

The same situation applies to the removal of all gross accumulations upon the exposed tooth surfaces before subgingival curettage. Drs. Bernard Friedman and Johnson stress the importance of this step very early in treatment. This should be done without injury to the gingivæ and without discomfort to the patient. If one includes the area immediately underneath the marginal gingiva and without any injury to it, the many advantages of applying this step at the beginning of treatment are multiplied. The pa-

tient in 90 per cent of the cases is given some relief and is instantly grateful to the operator. He should be allowed to watch the operation where the mouth has been least well cared for, in a magnifying hand mirror. He will be shocked at his neglect and will agree to the need for improvement in his methods of home care. It gives the periodontist his best opportunity for dental educational efforts and a chance for dentist-patient acquaintance and adjustment. After this treatment, if followed by the frequent use of a mild warm saline solution, the gingival tissues will be less liable to injury from new methods of home care and new equipment. There will be less bleeding from subgingival curettage, and a much more agreeable field in which to operate for the periodontist, regardless of the method used. Healing is more rapid and there is less discomfort for the patient during the course of treatment. The time required for completing the case is reduced. The patient can himself note immediate improvement.

There is a difference of opinion also as regards the time to relieve occlusal stress. Dr. McCall believes it should be done earlier in the procedure of treatment than do some of the Discussors and Dr. Leonard. It seems that this should be settled by the individual case requirements. There is no doubt that in many instances teeth are extruded from their sockets where there is much inflammation thereby causing occlusal traumatism. In such a case it would be advisable to wait until the inflammation has subsided after curettage, before adjusting the occlusion. On the other hand, if the injury is caused primarily by severe maladjustment of occlusion, then immediate relief of this is mandatory. In other words, the chief etiologic factor should be the first to be removed.

While much of the "Discussion" deals with *methods* of practice rather than steps in procedure, such as the use of auxiliary assistants in periodontia, periods for recalls, how long the teeth should last after treatment, who is most competent to care for the "old patient," etc., nevertheless, it is all interesting including the mention of omissions from the "Initial Statement." The use of oxygen in treatment was missed by Dr. Smith and should have been placed in the schedule of treatment. Neither is the use of paraffin and surgical cement packs mentioned, as to when used, although many periodontists who practice conservative methods of treatment use them constantly. Neither is sufficient mention made of the position and importance of the subject of nutrition in the plan for treatment. Dr. Rowe Smith suggests the use of the intraoral kodachrome photographs in addition to radiographs for recording conditions.

It is only by measuring personal opinions with those of others that they can be properly evaluated. Especially for this reason is the "In Our Opinion" Department valuable.

—G.R.S.

ANNOUNCEMENT

THE OFFICERS and the Council of the American Academy of Periodontology decided that, owing to the inconveniences of travel and also because of the lack of hotel accommodations, it would be unwise to attempt to hold an annual meeting in 1945, even though travel restrictions have been lifted. Instead, a reunion meeting is being planned and will be held in 1946, immediately preceding the next annual meeting of the American Dental Association, at the place selected by that organization for its meeting.

SAMUEL R. PARKS, *President*
RAYMOND E. JOHNSON, *Secretary*

Editorial

Salesmanship and Periodontia

WE ARE continuously subjected, to high pressure salesmanship from various sources, from the radio, advertisements in the daily press and in current magazines, and the many sales agencies. We are thus kept informed, according to the salesmen, of the advantages of spending our income for this or that article or gadget. Dental patients are made "pink tooth brush" conscious and get most of their advice on dentifrices from radio announcers. "Selected", "tested", "glamorous", "conversation pieces", "good to the last "drop", "regal", and hundreds of other terms have become bromidic from repetition.

While as members of a profession, we have not been taught much, if anything, about salesmanship, the public is being educated to expect much of their information on all subjects, the easy way, rather than thinking things out for themselves or making a personal effort to get it. Grand Opera and good orchestral music is now being sold the public over the radio to counteract the crooners and jazz band music. We have been sold War Bonds and Rationing. Our children should be sold on going to school and on the advantages of education with as much persistence as they are sold on jitter-bugging.

It was stated by the Sales Manager of a large automobile company, that the basic principle of salesmanship was to make people dissatisfied with what they had in the particular line to be sold to them. Could it be that many dental patients have been made dissatisfied with their natural teeth, and that artificial dentures have in this way been sold to some who could have saved their own teeth with greater comfort and satisfaction? It is granted that for hundreds of thousands, artificial teeth seems to be the only solution, however, the habit of recommending extractions can become a fixed one, as well as a point of view.

Every dentist, whether he admits it or not, must sell himself to a new patient by means of his knowledge of dentistry, his honesty, skill, personality and his consideration for their best interest. He retains patients in his practice by the same means. Few come for the first appointment with unqualified confidence in the dentist. This confidence must be won and kept if a stable practice is desired. A dental patient will place the same value on his teeth that he has been educated to estimate, by his dentists, past and present. If the extraction of two or more molars, without good and sufficient reason, means nothing to the dentist, it generally will mean but little to the patient at the time. Later, he may realize how much he has lost because of a masticatory handicap, or the loosening of other teeth from the extra strain placed upon them.

Health measures should be sold to the public with the same insistence that remedies are sold by radio. We have an unusual opportunity in dentistry to *sell* patients on, and to educate them in oral health and preventive measures, in the

value of the natural teeth *and* their supporting tissues, and in their personal accountability for home care.

We have already sold them on full dentures, gold inlays, "X-rays", the possible relation of infected teeth to arthritis and other physical ills, extractions, and orthodontia, with telling effect. Why not with equal enthusiasm sell them periodontia, dentistry for children, oral health, preventive measures of all kinds, health of the supporting tissues of the teeth, good nutrition, oral hygiene, and their personal responsibility in keeping the teeth and interproximal spaces *clean*, and above all, sell them natural teeth in a healthy environment.

Too many important teeth are carelessly sacrificed, in fact complete natural dentures are often condemned because radiograms show moderate alveolar resorption. Every periodontist will endorse the idea of more conservatism in condemning teeth for extraction and an improved evaluation placed upon them. Let us avoid making patients dissatisfied with their own teeth by making these more comfortable and attractive and retain for co-operative patients, all savable teeth. In short—*sell* the patient's own teeth to him and educate him in their care and preservation. Too many people expect to lose their teeth during middle life instead of expecting them to last as long as they are needed, and for this reason alone they often neglect them for years.

The first president of the American Academy of Periodontology, Dr. Austin F. James, thirty-nine years ago, proposed a motto for the society—"Real Teeth". May we, therefore, continue to keep constantly in mind—*real teeth* and sell them to our patients. —G.R.S.

In Memoriam

CHARLES A. WALL

WILLIAM A. JAQUETTE

JULY 3, 1945

(Continued from 16(2):75. April, 1945)

(a) Periodontal Histopathology

210—Glickman, I., Morse, A., and Robinson, L.: **The systemic influence upon bone in periodontoclasia.** *J.A.D.A.* **31**:1435. Nov. 1944.

A series of animal experiments were conducted to determine what effects an alteration of the systemic background would bring about in bone subjacent to areas of gingival inflammation. It was found that the bone of the jaw undergoes changes similar to those which occur in other bones as the result of generalized skeletal disturbances of systemic origin. Alveolar bone loss may occur in periodontoclasia as a result of generalized skeletal disturbances in the absence of gingival inflammation. Also, the authors found that inflammation in the gingival crevice may result in bone resorption and reduction in the height of the interdental septa in the absence of generalized skeletal disturbance. The severity of bone loss subjacent to gingival inflammation is increased by generalized skeletal disturbances of systemic origin.

—*H. M. Goldman*

211—Goldman, H. M.: **Marginal periodontitis.** *Am. J. of Ortho. and Oral Surg.* **31**:93, 1945.

Marginal periodontitis is a disease of the marginal periodontium characterized by inflammation and resorption of the alveolar crest. Clinically it must be differentiated from gingivitis and senile atrophy. Resorption of the alveolar crest takes place medially, first the supporting bone being destroyed and then the lamina dura. This results in a cuplike notch in the alveolar crest, a finding which can be correlated in the radiographic examination of this disease. Although marginal periodontitis is associated with calculus in the gingival crevice and inflammation of the gingiva, study of human autopsy material reveals that neither the depth of the gingival crevice nor the degree of the inflammation bear any relationship to the condition of the alveolar crest. Why marked resorption is seen in some cases and not in others cannot be established from histologic evidence.

—*Author's Abstract*

212—Goldman, H. M.: **The relationship of the epithelial attachment to the adjacent fibers of the periodontal membrane.** *J. D. Res.* **23**:177. June, 1944.

A fundamental process pertaining to the formation of the pathologic gingival sulcus is the apical proliferation of the epithelial attachment alongside the cementum of the tooth. The author believes that this apical migration is dependent upon the maintenance of the insertion of the principal fibers of the periodontal membrane which lie in apposition to the epithelium. Epithelial proliferation can take place only through the destruction of these connective tissue fibers.

—*Author's Abstract*

(e) Symptomatology and Diagnosis

(Continued from 16(2):73. April, 1945)

377—Erger, B. D.: **Erythema multiforme pluriorificialis.** *Military Surgeon* **98**: 307, 1944.

A case of erythema multiforme pluriorificialis is presented which is similar to those reported by Stevens and Johnson, Ginandes, Ageloff, and many others. This syndrome is more commonly found in Europe than in America, but seems to be on the increase in this country. It is characterized by an abrupt onset with fever, conjunctivitis, stomatitis, and cutaneous lesions. A history of exposure preceding the onset is thus far lacking. The disease runs a more or less protracted course with a prolonged convalescence with visual impairment, if not complete blindness, as an almost constant sequel. Numerous degrees of eye involvement have been reported, ranging from simple catarrhal conjunctivitis to ocular destruction. Buccal involvement too may vary from a mere troublesome stomatitis to a profoundly severe, malodorous inflammatory process of the lips, gums, tongue, buccal mucosa, and pharynx, with copious salivation and dysphagia. These lesions usually heal slowly, but completely. The

cutaneous lesions are macular, papular, vesicular, and bullous, and are rather more likely to be uniform than multiform, but otherwise resemble erythema multiforme more closely than any other dermatosis. Erythema multiforme was the first diagnosis that suggested itself, in the present case, despite the atypical distribution. Differentiation from sepsis, toxicoderma, syphilis, and pemphigus depends upon the history, the appearance, character, and course of the eruption, and negative laboratory data. The eruption, which may appear on any portion of the body surface excepting the scalp, progresses for about seven to ten days and then begins to crust, scale, and fade, leaving pigmented spots which become progressively paler in color. No pitting or scarring has been observed.

The evidence gathered thus far is not sufficient to support the contention that it is a new infectious disease, or even an infectious disease. Until further investigation discloses the causative agent or factors it is suggested that it were best to consider the condition one of severe, atypical erythema multiforme with grave prognosis for the eyes.

—*H.M.G.*

378—Esterberg, H. L., and White, P. H.: **Sodium dilantin gingival hyperplasia.** *J.A.D.A.* **32**:16. Jan. 1945.

The authors observed 244 patients over a period of nearly three years. The youngest patient was 3, the oldest 69 years. Dilantin hyperplasia occurs in 54 per cent of all persons taking the drug. The most severe reactions occurred between the ages of 15 and 30. Not age, sex, length of treatment or size of dosage, but rather the individual tolerance or sensitivity to the drug, determines the incidence of gingival reaction. Local conditions, open bite excepted, have little influence on the incidence or severity of the oral reaction.

The initial change consists of a slight inflammation of the interdental papilla, followed by a pinhead tissue extrusion. This is followed by other areas becoming small hypertrophied bulbs, almost pendulous and very often bleeding easily on brushing or eating. Later the mass becomes a pendulated, lobulated papilla. By the time this stage is reached, most patients complain of pain. As the hyperplasia increases it remains circumscribed to the labial or buccal surfaces of the teeth. In some cases, the lobulated character persists, the teeth showing through a cleft in the tissue. The normal level of the gingival attachment to the tooth was not disturbed.

An intensive daily routine of vigorous massage with toothbrush and gum stimulators can check the gingival overgrowth in many cases. For the remainder and those unable to follow the massage, only repeated surgical measures or complete cessation of the drug therapy will remove the excess tissue.

—*H.M.G.*

379—Levy, B. M.: **Oral manifestations of histoplasmosis.** *J. A. D. A.* **32**:215, 1945.

Histoplasmosis is a fatal fungous disease first described in 1905. Since that time seventy cases have been described of which 20 per cent had oral lesions. Histoplasmosis is characterized clinically by a low grade, irregular pyrexia, splenomegaly, hepatomegaly, and lymphadenopathy. Anemia and leukopenia are observed in most cases. Ulcerated granulomatous lesions of the skin, oral mucous membranes, pharynx, and gastro-intestinal tract occur. The disease can be localized or systemic and may run an acute or a chronic course, lasting from a few weeks to many years.

Histoplasmosis of the oral cavity may involve any or all of the structures present. Nodular ulcerative or vegetative lesions on the tongue, lips, buccal mucosa and hard and soft palate are suggestive. The buccal mucosa is most often involved by an ulcerative process which spreads over the gums. These ulcers are usually covered with a gray or pinkish membrane, very similar to the membrane found in many ulcerative processes in the mouth.

Since there is no method of diagnosing histoplasmosis other than demonstration of the organisms in tissue microscopically or isolation of the organism on culture media, it is well to keep the possibility of this fungous disease in mind whenever we are faced with a chronic granulomatous process in the mouth.

—*H.M.G.*

BOOK REVIEW

Periodontal Diseases and Soft Tissue Lesions of the Oral Cavity. By Arthur H. Merritt, M.S.,D.D.S.,Sc.D.,F.A.A.P. *Third Edition*—New York, The Macmillan Company, 1945. 258 pp., 53 illustrations.

On June 13, 1945, the author of this book was given the honorary degree of Doctor of Science by New York University before a Commencement Assembly numbering thirteen thousand, composed of graduates, faculty members, and guests. He is a Past President of the American Dental Association, and also of the American Academy of Periodontology, and his professional attainments are such as to place him in the position of a leader among leaders in our profession. This book should be widely read, not only because the author is an incomparable arbitrator in any matter of controversy pertaining to the practice of dentistry, but also because the book itself is of a particularly high order.

This treatise has been written for the busy practitioner who has to face periodontal problems in daily practice and wishes to be told in the most concise manner possible just how to deal with them. If the author doesn't know the answer he is big enough to say so and does say so.

The book is in two parts. Part One is the same as the previous editions except that it has been brought up to date and covers such subjects as Gum Recession, Gingivitis, Vincent's Infection, Etiology and Treatment of Periodontoclasia, Prevention of Periodontal Disease, Diet, Sensitive Teeth, and Splinting Teeth.

Part Two is entirely new and deals with soft tissue lesions of the oral cavity. A considerable number of systemic diseases produce lesions of the mouth including the periodontal tissues and as the author points out, this is true of avitaminosis, blood dyscrasias, systemic infections, sensitivity to certain drugs etc., all of which exhibit symptoms in the mouth. The following are discussed: Pigmentation, Avitaminosis, Perléche, Herpes Simplex, Tuberculosis, Syphilis, Agranulocytosis, Leukemia, Purpura, Pernicious Anæmia, Lichen Planus, Pemphigus, Cancer, and Stomatitis from drug ingestion.

In these days it is particularly important that a dentist should be able to make a differential diagnosis of these diseases and direct the patient into proper hands. In those cases which should be treated by the dentist, the author states clearly his method of treatment and does so in a clear, concise manner that anyone can understand.

In perusing this book we find such statements as these: "More teeth are lost from periodontoclasia than from any other dental disease": "The disposition to regard the grinding of teeth as the *sine qua non* of treatment is less in evidence than it was a few years ago": "We should warn against the mutilation of teeth by excessive grinding": "To tell patients that Vincent's infection is highly contagious and to warn them against infecting other members of the family, in the light of present knowledge, would seem to be unwise": "The family dentist should be first to detect signs of gum recession and take such steps as may be necessary to prevent its further progress": "In the light of present experience it would seem that electrocoagulation has little or no place in the treatment of periodontoclasia": "Just how large a part diet plays in the cause and treatment of periodontal disease is a question which at present no one is prepared to answer": "There was never a case of periodontoclasia that reached an advanced stage that could not at the beginning, have been cured at an infinitesimal cost."

Who can disagree with the above sane statements and very many others equally helpful? Following gum resection the author uses a surgical paste mixed stiff and rolled into cone shaped pellets and pushed between each proximal space, and with several changes allows it to remain there three to four weeks. This reviewer does not make a thick mix but rather a soft mix, places

the same with the spatula on the surface operated upon, and moulds into position by pressing the lip or cheek against the same, leaving on for not more than one week, as a rule, and only changing or replacing if soreness exists, which is seldom. This does not mean that the practice adopted by the author is not the ideal.

Merritt uses 5% chromic acid in the treatment of Vincent's infection: Fish of London uses 20%: this reviewer uses 10%. However, that does not mean that 5% is not strong enough. Whoever heard of periodontists agreeing on everything! They cannot even agree as to the ideal way to brush the teeth.

This book has been perused with unusual pleasure and profit and because there is no one better able from scholastic attainment, clinical study, and years of experience, to teach this subject, one has no hesitation in urging the general practitioners of dentistry to study it most carefully.

—M. H. GARVIN

INTERESTING LETTER RECEIVED FROM A *Non-resident Member* AMERICAN ACADEMY OF PERIODONTOLOGY BY DR. DONALD MOSHER, *Member*.

Copy

Dear Dr. Mosher:

. . . I gave up practice some two and one-half years ago and joined the Royal Australian Air Force, so consequently I have been moving about the world a bit.

I was made periodontal specialist to the Air Force and am now Senior Periodontist in the service with the rank of Squadron Leader which corresponds to your Major . . .

I have seen a lot of your fellow countrymen out here and was associated closely for some time with a group from Wisconsin.

I am now stationed in Melbourne which is the Air Force Headquarters and it looks as though I will be there till the end of the war though I travel all over the country giving post-graduate courses and clinics to service and civilian dentists.

I am also in the middle of compiling a text-book *Periodontal Treatment for the General Practitioner* which will be published by the Air Force so when it is ready I will send you a copy.

A bundle of literature has just arrived from the American Academy of Periodontology.

I'm afraid I have been a very poor correspondent in the past, but letters have sometimes taken months to find me, but now things are stabilized and I can resume a normal existence again.

My address now is:

Squadron Leader George Christensen
R. A. A. F. Directorate of Dental Services
Spring St.
Melbourne, Victoria, Australia.

I would be very grateful to learn of anything new in the periodontal world and I will also send you a reprint of the work we have done on Penicillin in the treatment of "Acute Ulcerative Gingivitis" as soon as it is published.

With the very best wishes to you all. . . .

Yours fraternally,
GEORGE CHRISTENSEN

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